SEGUIN. (E.C.)

A SECOND CLINICAL STUDY

OF

HEMIANOPSIA.

CASES OF CHIASM-LESION. DEMONSTRATION OF HEMIOPIC PUPILLARY INACTION.

BY

E. C. SEGUIN, M.D.,

Corresponding Member of the Société de Biologie of Paris, of the Verein für innere Medicin of Berlin, Honorary Member of the Société Anatomique of Paris, etc.

presented by the author

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A SECOND CLINICAL STUDY OF HEMIANOPSIA. CASES OF CHIASM-LESION. DEMONSTRATION OF HEMIOPIC PUPILLARY INACTION.[1]

By E. C. SEGUIN, M.D.

I BEG leave to present to the Society notes of three cases of hemianopsia of the peripheral or neural form, probably caused by a lesion of the optic chiasm. None of these cases are completed by autopsy, yet I think that the diagnosis is clear enough to render them worthy of record, and to make them the subject of a few clinical and diagnostic remarks.

All of these cases present the remarkable pupillary light reaction, first indicated by Von Graefe, and designated by Wernicke as hemiopic pupillary reaction. One of the patients is present this evening, and upon him I may be able to demonstrate this rare and valuable symptom, which, to the best of my knowledge, has not yet been observed (or at least recorded) in this country.

I shall append a series of diagnostic propositions bearing upon all varieties of hemianopsia, which, though far from perfect, may perhaps prove useful for our further study of this symptom.

CASE I.—*Loss of vision in left eye; temporal hemianopsia with right eye. Complete atrophy of left optic nerve; partial of the right. No other symptoms of cerebral lesion. Hemiopic pupillary inaction.*

H. S., a grocer's clerk, a German, 20 years of age, presented himself at the Manhattan Eye and Ear Hospital, service of Dr. David Webster, on October 26th, 1887. Dr. Webster kindly referred him to my department for observation and treatment.

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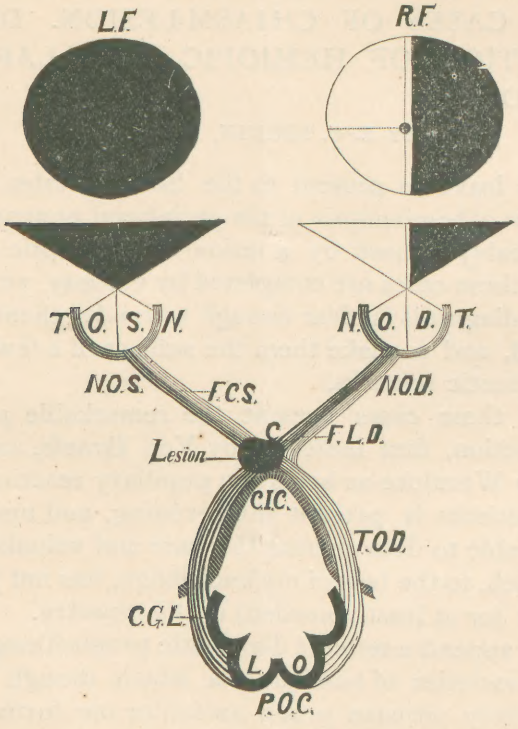
[1] Read before the New York Neurological Society at the stated meeting held Dec. 6th, 1887.

Health has been fairly good, except that for six years he has had occasional attacks of bilateral headache, mostly frontal, often accompanied by nausea and vomiting. This was apparently migraine. Headache not in family. In the last two years more constant frontal pain. No injury or syphilis. Two years ago, the patient accidentally discovered that his left eye was partly blind; he could see only large objects. This has since progressed to total loss of vision. In the last few months less headache, without vomit-



LF, left visual field; RF, right visual field; OS, left eye; OD, right eye; N nasal; T, temporal halves of eyes; NOS, left optic nerve; NOD, right optic nerve; FCS, left fasciculus cruciatus; NLD, right fasciculus cruciatus; C, chiasm; TOD, right optic tract; CIC, commissura inferior cerebri (non-optic fibres of chiasm); CGL, corpus geniculatum laterale; LO, lobus opticus, which together make up POC, the primary optic centres. For central parts of optic apparatus cf. fig. on p. 35 of this JOURNAL for Dec., 1886.

ing. No other symptoms, sensory or motor. States that he smells and tastes well. Dr. Webster found L. V = o; R. V = $\frac{20}{200}$. The right visual field showed temporal hemianopsia, as represented in above diagram

The left optic nerve is fully atrophied, the right partly. Examination shows that neither pupil responds when light is thrown into the (blind) left eye. When the light is thrown into the right eye, contraction occurs in both pupils ; showing that centripetal conduction is preserved in the right optic nerve to the lobus opticus, and that centrifugal conduction is perfect through both motor oculi nerves to irides.

The right pupil apparently reacts well to light. As usual, the vertical division line between the light and dark half-fields of that eye falls outside of the point of fixation. No paralysis of ocular muscles or of any part of the body. In short, there are no other symptoms of cerebral or spinal disease. General health fairly good.

Dr. Webster and I considered the case as one of chiasm-lesion, involving three-fourths of the decussation, *i. e.*, destroying both fasciculi of the left optic nerve and the fasciculus cruciatus of the right eye. That the lesion is in the peripheral or basal part of the optic apparatus is indicated by the loss of pupillary reflex in the blind left eye, and by the extreme degree of atrophy present. By the help of the above diagram of the decussation the probable seat of the lesion is made clear.

That the lesion does not involve one optic tract caudad of the chiasm is certain, because in that case there would be lateral or homonymous hemianopsia in both eyes.

In this case, a search was made for hemiopic pupillary inaction in the right eye, with the following results, which I demonstrated to Dr. Webster and a number of the staff of the hospital, and some other physicians. When the light from an ophthalmoscopic mirror was thrown directly into the right pupil, a good reaction was at once obtained in the two irides.

When the beam of light was made to strike the cornea from the nasal side (the side with vision) at any angle, a reaction was also obtained all around the pupil in both eyes.

When, however, the beam of light was made to enter the right eye from the temporal or dark side at an angle of 60°, or rather less, striking chiefly the nasal or anæsthetic half of the retina, no reaction, or almost none, occurred in the left pupil (and in the right). Therefore, we had before us a striking example of hemiopic pupillary reaction, or *inaction*, as I would prefer to call it. An interesting fact is that the entire iris responds to the stimulus applied to the temporal half of the retina and only transmitted centripetally by the fasciculus lateralis of the right eye.

That the whole muscular apparatus of the iris (sphincter or constrictor pupillæ) should contract from a reflex action originating in one half of the retina is easily understood if we remember that the ciliary nerves go to form a plexus containing ganglion cells in the iris. With such a nervo-muscular apparatus, the motor impulse must of necessity be diffused throughout the entire iris, and not restricted to one of its halves, or a smaller part.

November 21st.—Re-examined for hemiopic pupillary inaction. Same result obtained, viz., when the beam of light is thrown into the right pupil from any angle on the nasal side, or from 90° directly into the centre of the retina, or to the temporal (blind) side as far as 70° or 60° on an equatorial arc, a good pupillary reaction is obtained. When, however, the ray of light is made to enter more obliquely from the temporal side, from 60° to 40° on the equatorial arc, no reaction was obtained in the eye examined or in the left blind eye.

Both pupils contract under accommodative effort.

L. V = 0; R. V = $\frac{6}{200}$. With the right eye can just make out No. XIV. Jaeger at 8 inches.

CASE II.—*Partial blindness, with marked atrophy of both optic nerves. Bi-temporal hemianopsia, plus obscuration of upper nasal quadrant of left field. No other symptoms of cerebral disease. Hemiopic pupillary inaction in right eye.*

J. H., a male, aged 41 years, was referred from the ophthalmic department (service of Dr. D. Webster) to the nervous department of the Manhattan E. and E. Hospital as an interesting case of atrophy of optic nerves probably due to intra-cranial disease.

The patient stated that since the spring of 1886 there has been steadily increasing failure of vision in the left eye, to almost complete blindness. Occasional frontal headaches, never very severe. Has had diplopia at times, which he describes quite intelligently, in a rather peculiar way, stating that where there were two objects he would see three, where there were four he would see five. For several years occasional slight attacks of vertigo. No other symptoms indicating cerebral or spinal disease.

Denies syphilis and injury to head.

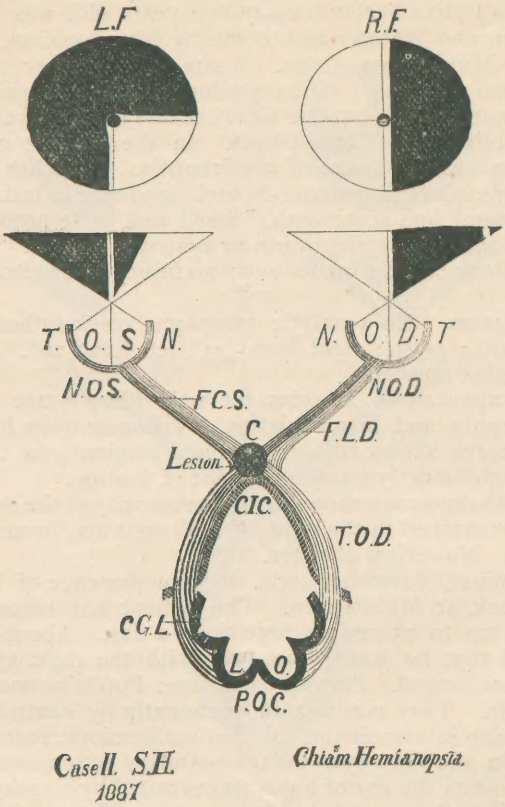
Examination shows: Ocular muscles normal. Pupils moderately dilated and responsive to light. Both optic nerves appear considerably atrophied. Visual fields, as per subjoined diagram (blackboard test).

This peculiar form of hemianopsia (nearly typically bi-temporal) would indicate the existence of a lesion so placed as to destroy both fasciculi cruciati, and the basal or inferior half of the fasciculus lateralis of the left eye.

Nov. 11th. Re-examined. Fields (tested on blackboard) are as before, except that in the field of the right eye the vertical line of separation now passes through the point of fixation. Both pupils contract with accommodative effort. Both pupils independently show reaction to light; barely perceptible in left eye, marked in right eye; slight but distinct transferred or consensual light reaction from one eye to the other. Right eye exhibits hemiopic pupillary inaction. When light is thrown into the pupil from the temporal side up to 60° no reaction whatever takes place; but when the light pencil is thrown in from the nasal side at any angle and past the optical axis (90°) to about 60° on the temporal side, distinct iridic contraction occurs. The reaction is obtained

equally well in these meridians from any degree above or below the equatorial line.

Left pupil reacts slightly to central illumination, but tests on either side yield no trustworthy or definite reactions.[1]



Case II. S.H. 1887 *Chiasm Hemianopsia.*

LF, left visual field; RF, right visual field; OS, left eye; OD, right eye; N and T, nasa and temporal halves of each retina; NOS, left optic nerve; NOD, right optic nerve; FCS, left fasciculus cruciatus; FLD, right fasciculus lateralis; C, chiasm; CIC, commissura inferior cerebri (non-optic fibres of chiasm); TOD, right tractus opticus; CGL, corpus geniculatum laterale; LO, lobus opticus or anterior group of corpora quadrigemina, which together make up POC, the primary optic centres.

[1] Re-examined Dec. 3d. Condition as before except that the optic nerve atrophy has progressed, and is more evident in right nerve. The right visual field is exactly hemianopsic; the left field shows a further geometric loss; one-third (the inferior) of the lower nasal quadrant now being dark. The pupils are larger and nearly inactive. A faint but distinct hemiopic pupillary inaction is obtained when the right eye is tested as above described. V. R.$=\frac{8}{200}$, V. L.$=\frac{20}{50}$.

This result is interesting as showing that the geometric amount of field-reduction is not the only factor in loss of vision.

CASE III.—*Almost total loss of vision in the right eye. Temporal hemianopsia with left eye. Partial atrophy of both optic nerves. Distinct hemiopic pupillary inaction. No symptoms of cerebral disease.*

A. C., an Italian barber, twenty-five years old, was referred to Drs. Agnew and Webster and to me by his physician, Dr. Rufus Baker, of Middletown, Conn. I saw him October 10th, 1887.

Some two years ago vision gradually became impaired. The left eye improved (?), but the right became progressively worse to complete blindness. No diplopia. In the last two months the patient has become aware of hemianopsia. No other symptoms, except some bilateral temporo-frontal headache in last two weeks only. General loss of strength. Smell and taste preserved; no hallucinations. No epileptiform or syncopal attacks.

No etiology can be made out; no injury to head or syphilitic infection.

Examination. No paralytic symptoms or anæsthesia. Equilibrium good; grasp: right hand 23°, left hand 20°. No ataxia. Patellar reflex normal.

Ocular apparatus. Muscles act well. Pupils are equal and active, directly and in association. Vision is quite lost in right eye. Left eye shows regular temporal hemianopsia, the vertical line passing distinctly outside of point of fixation.

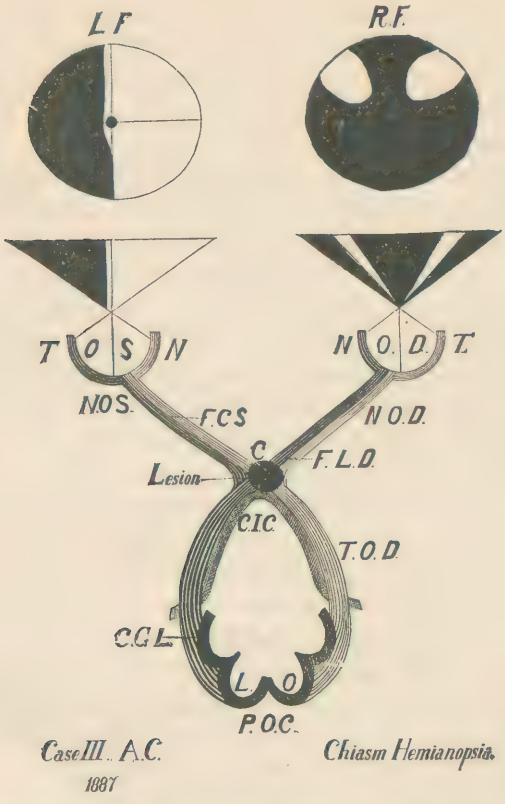
The ophthalmoscope shows moderate atrophy of the optic nerves, much more marked in the right eye. Vessels are, however, full in both eyes. No retinal changes.

Re-examined November 20th, 1887, in presence of Drs. Baker and Hallock, at Middletown. The patient has taken iodide of potassium up to 75 grains three times a day. About two weeks ago found that he had some sight with the right eye upward. Left eye unchanged. Present condition: Pupils normal, medium and mobile. They contract independently by central illumination, and also in association. No distinct hemiopic inaction can be obtained in the right eye, though pupillary action seems quicker and fuller when the ray of light concentrated by a concave mirror is thrown into the pupil from above on the right or left of vertical meridian. In the left eye, typical hemiopic pupillary inaction is obtained: *i. e.*, when light is thrown obliquely into pupil from the temporal or dark half-field no reaction occurs. When the ray of light is brought nearly (at 60° or 70°) in the pupillary axis, in it, or carried beyond into the nasal half-field, a full and quick pupillary reaction occurs.

Vision. With the right eye a large white object, such as one's hand, or a piece of paper, is perceived in a part of the two upper quadrants of the field. The two quadrants are separated by a dark strip vertically placed in the field, as shown in annexed diagram. In the left eye there is temporal hemianopsia as before, with vertical line (not quite straight this time) passing outside of

the point of fixation. Central vision is very good; patient can read No. I. Jaeger at 7 inches.

The ophthalmoscope shows as before whitish nerves with remarkably good-sized vessels; the right optic nerve is very white.



LF, left visual field; RF, right visual field; OS, left eye; OD, right eye; N and T, nasal and temporal halves of each retina; NOS, left optic nerve; NOD, right optic nerve; FCS, left fasciculus cruciatus; FLD, right fasciculus lateralis; C, chiasm; CIC, commissura inferior cerebri (non-optic fibres of chiasm); TOD, right tractus opticus; CGL, corpus geniculatum laterale; LO, lobus opticus, or anterior group of corpora quadrigemina which together go to make up POC, the primary optic centres.

It is evident from the form of the hemianopsia in these cases, and from the early appearance of atrophy of the optic nerve without choked disk, that they belong to the group of peripheral hemianopsia due to a basal or neural lesion, as distinguished from that other group which, on a former occasion, I brought to the attention of the Society,

in which the hemianopsia is produced by a central or cerebral lesion, situated anywhere in the optic apparatus caudad of the primary optic centres.

That the peripheral lesion in the cases here related is not one affecting one optic tract is shown by the form of H., *i. e.*, in its not being of the lateral or homonymous form. The lesion is quite surely at the chiasm itself, situated as approximately represented in the diagrams which accompany each case.

There are several points of interest in the semeiology of these cases, but I shall occupy the time of the Society only by the consideration of one symptom presented by all three patients—a relatively new symptom, and one which I believe has great diagnostic value, enabling us to decide with nearly positive exactness whether a hemianopsia is due to a central or to a peripheral lesion. I refer to the symptom called by Wernicke [1] hemiopic pupillary reaction, and which I now propose to designate, more accurately perhaps, *hemiopic pupillary inaction.*

Allow me to clearly state the nature of the symptom and the conditions of its demonstration.

The normal optic apparatus contains a reflex arc that automatically regulates the size of the pupils under different degrees of illumination. The component parts of this arc are: the whole retinal expansion (with special sensitiveness of the macula) as a receptive organ for light, the optic nerves and tracts as centripetal channels of transportation of the impulse produced by light to the anterior group of the corpora quadrigemina (the lobi optici) where a reflection takes place to the nuclei of the motor oculi nerves (to both nerves at once). The mechanism and the physiology of this part of the action is unknown to us. From the nuclei of the motor oculi a centrifugal or motor

[1] C. Wernicke, "Ueber hemiopische Pupillenreaction." Fortschritte der Medicin, I. Heft 2, 1883.

I owe an apology to Professor Wernicke for having misquoted and perverted the true sense of his article in a brief reference which I made to it in my paper on the Pathology of Hemianopsia of Central Origin. It is incomprehensible to me now how I could have failed to comprehend Wernicke's paper on first reading it.

impulse goes out to the ciliary nerves and to the iris muscles, producing contraction. A luminous impulse from one retinal expansion produces the reflex motor act in both irides (associated or consensual pupillary reflex), and the entire iris contracts in all instances because the termination of the ciliary nerves in the irides is plexiform, with intercalated ganglion cells in the muscle.

If a ray of light be made to enter the eye directly in its optical centre, striking the macula lutea, the pupillary reaction is quickest and most complete. If now the light be so moved as to cause the ray to impinge from various angles above or below, nasalward or temporalward from the optical axis, pupillary reaction still occurs.

In pathological cases, the reflex arc may be broken at various points. (a) The entire retina or one of its halves, or a more limited part of it, may be insensitive to light through local disease or neural degeneration. (b) The conducting centripetal paths (optic nerves), may be interrupted by a lesion; in these categories belong our cases of hemianopsia with defective pupillary reaction. (c) The centre for the reflex act, the gray matter in which the mysterious transformation of a sensory into a motor impulse takes place, which, according to most recent researches (von Gudden and others) is in the lobi optici, may itself be diseased. (d) The conducting centrifugal paths (motor oculi and ciliary nerves) may be interrupted by a lesion, or the nuclei of the motor oculi may be diseased. (e) The terminal motor organ, the iris, may be rendered rigid or inactive by local disease (iritis). This general statement will serve to explain all varieties (pathologically speaking) of diminution or loss of pupillary reflex.

In the variety to which I desire to ask your attention this evening, viz., *hemiopic pupillary inaction*, one-half of each retina being physiologically inert, or anæsthetic, fails to receive any impulse from the light which is thrown on it. All three cases related, including the patient upon whom I shall attempt a demonstration, present this symptom.

But certain restrictions must be placed upon the meaning of the word hemiopic as applied to the results of testing, for we find the facts as follows in these cases:

If, in the hemiopic eye, we throw the pencil of light (concentrated from a lamp by an ophthalmoscopic mirror) directly into the eye in its optical axis, we obtain a full and quick pupillary reaction. If, now, we move the mirror more and more nasalward from the optical axis, and throw the beam of light through the pupil upon the nor-

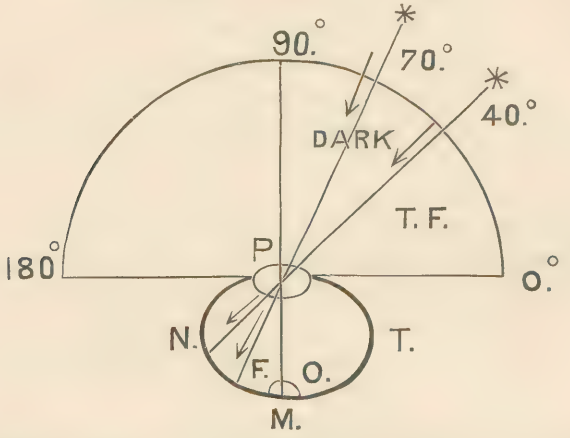


FIG. 4.—Diagram illustrative of test for *hemiopic pupillary inaction*. The lines represent a horizontal plane through the left eye and its visual field. FO, fundus oculi; M, macula lutea; N, nasal half of field which is anæsthetic in temporal hemianopsia; T, temporal half of retina; P, pupillary aperture. 180° to 0°, the equatorial arc or semicircle. 90°, vertical point and line passing through centre of eye to M. 70° and 40°, rays of light striking the insensitive nasal half of retina, producing no pupillary reflex. About 60° is a fair average for the angle obtained in the various tests of the three cases.

mal (temporal) half of the retina, a good reaction occurs until the light ceases to enter the pupil. If we next move the mirror outward or temporalward from the optical axis of the eye, we obtain pupillary reaction over quite an arc of the circle; but after passing a point between 60 or 40 degrees from the horizontal pupil line (see Fig. 4), pupillary reaction no longer takes place, and we have a demonstration of hemiopic pupillary inaction. On first thought, it would seem fair to conclude from this experiment that only the peripheral 40 or 60 per cent of the

nasal half of the retina is insensitive or anæsthetic, but this is not so. The reason why the light experiment is not as geometrically exact as the perimetric test is, that it is mechanically impossible to throw a beam of light so that it shall be focussed only on very limited portions of the retina. Even with a small pencil of light there is more or less illumination of the entire pupillary aperture and diffusion of light within the eye. If we could exactly restrict the action of the ray of light, the pupillary immobility would be evident the moment the mirror was so displaced temporalward (in temporal hemianopsia) as to strike the retina a little to the nasal side of the macula.

It follows that in testing for hemiopic pupillary inaction we must observe a number of precautions. My own plan of procedure is as follows: The patient being in a dark or nearly dark room, with the lamp or gas light behind his head in the usual position, I bid him look over to the other side of the room, so as to exclude accommodative iris movements (which are not necessarily associated with the reflex). Then I throw a faint light from a plane mirror or from a large concave mirror held well out of focus, upon the eye and note the size of the pupil. With my other hand I now throw a beam of light, focussed from the lamp by an ophthalmoscopic mirror, directly into the optical centre of the eye; then laterally in various positions, and also from above and below the equator of the eye, noting the reaction at all the angles of incidence of the ray of light. With a little practice this can be thoroughly done in a few moments. In testing the pupils for other purposes (in cases of tabes, dementia paralytica, etc.) by ordinary daylight or lamplight, it is also important to make the patient look at a distant object in order to relax accommodation, and thus exclude accommodative iris movements.

Accommodative pupillary movements were preserved in all three cases.

The reasons why I have treated at such length of the symptom hemiopic pupillary inaction are: its intrinsic value for diagnosis, as will be shown farther on; and the

fact that, to the best of my knowledge, it has not been observed, or, at least, publicly referred to in this country.

In conclusion, I wish to append, as a sort of conclusion to my three papers on hemianopsia, a series of diagnostic propositions or laws, applicable to all cases presenting this symptom. It is far from my thoughts to claim that these propositions are final or absolutely exact in all particulars. They will doubtless be amended, but perhaps in their present shape they may temporarily prove useful to the practical physician.

FIRST CATEGORY OF CASES.

Vertical or quadrant hemianopsia alone; or, at least, without other special symptom of organic intra-cranial lesion.

1. Homonymous or lateral H. The lesion is either in the cuneus of the hemisphere opposite the dark half-fields, or it involves the tractus opticus opposite to the dark half-fields.

a. When the lesion is central, *i. e.*, involving the cuneus and adjacent basal gyrus, the optic nerves appear normal to the ophthalmoscope, central vision is good and the pupillary reflex is fully preserved. Except: When the lesion is a very large tumor, there may be neuro-retinitis, and, after a long time, atrophy of both optic nerves, with blindness and total loss of pupillary reflex. At no stage of the disease is hemiopic pupillary inaction observed.

b. When the lesion involves the tractus opticus, there is, from an early period, hemiopic pupillary inaction and partial optic nerve atrophy. Later in the course of the disease, neuro-retinitis of both eyes may occur, and be followed by atrophy, total blindness, and by pupillary immobility.

c. Homonymous quadrant obscuration. If, for example, the lower lateral quadrant of each visual field be obscured, the lesion involves only the basal half of the cuneus of the opposite side. (This proposition is justified by Hun's case, vide *The American Journal of the Medical*

Sciences, Jan., 1887, pp. 141–144). In such a case there would be full pupillary reaction.

II. Heteronymous H. is present, usually, with hemiopic pupillary inaction, and atrophy of the optic nerves.

a. Bi-temporal H. in both eyes; *i. e.*, the temporal or external half of the visual field of each eye is obscured: optic nerve atrophy and hemiopic pupillary inaction are present. The lesion is at the chiasm, in its median part, so placed as to destroy both fasciculi cruciati.

α—There may be temporal H. in one eye, and complete loss of vision with atrophy of the optic nerve and pupillary immobility in the other eye. The lesion is at the chiasm, so placed as to destroy both fasciculi cruciati and one fasciculus lateralis (Case I.).

β—There may be lateral H. of one eye and obscuration of three quadrants of the field of the other eye. The lesion is then at the chiasm, so situated as to destroy both fasciculi cruciati, and the ventral or dorsal half of one fasciculus lateralis (Case II.).

γ—With lateral H. of one eye, there may be a superior, partial, or quadrant obscuration of the other eye, as in Case III. at the last examination. In such a case, or in a similar one of irregular quadrant obscuration (not strictly lateral or homonymous), the lesion is an infiltrating one, affecting various bundles of the chiasm and adjacent optic nerves in an irregular way.

b. Bi-nasal H. is exceedingly rare. To my knowledge there is only one case on record with a post-mortem examination. There should also be in this form hemiopic pupillary inaction, and atrophy of the optic nerves. The lesion is at the chiasm, so placed as to injure both fasciculi laterales. In Knapp's case,[1] calcified and enlarged carotid arteries compressed the sides of the chiasm. A tumor might possibly so grow as to act in the same manner.

c. Superior or inferior H. This form is rarely due to a neural lesion. When present with a somewhat irreg-

[1] "Hemiopic and sector-like defects in the field of vision." Brown-Séquard and Seguin's Archives of Scientific and Practical Medicine, 1873, No. 4, page 308.

ular demarcation line, it is due to embolism of one of the main branches of the central artery of the retina. A lesion might theoretically be so placed at the chiasm as to injure its ventral or dorsal aspect only, thus causing the horizontal H., but we have no positive knowledge of such a condition.

d. Monocular H. of any form is due to a lesion of the optic nerve frontad of the chiasm, or to a lesion on one side of the chiasm. If not exactly geometric and without atrophy of the optic nerve, its cause is probably within the eye.

e. A few cases of rapidly varying forms of H. in the same subject have been described. A remarkable one has been recorded by H. D. Noyes and T. A. McBride.[1] The pathology of such cases is unknown; possibly, changing states of circulation and nutrition in the cuneus would explain the symptoms.

SECOND CATEGORY OF CASES.

Vertical or quadrant hemianopsia coinciding with other symptoms indicating definite cerebral disease.

I.—Lateral or homonymous H. in such combination.

a. Co-existing with hemianæsthesia and choreiform or ataxic movements of one-half of the body (of the limbs on the same side of the body as the dark half-fields), without marked hemiplegia, it is probably due to a lesion of the caudo-lateral part of the thalamus, or of the caudal division of the internal capsule on the side opposite to the dark half-fields.

b. Lateral H. with complete hemiplegia (spastic after a few weeks) and hemianæsthesia, is probably caused by an extensive lesion of the internal capsule in its knee and caudal part.

c. Lateral H. with common or typical hemiplegia (spastic after a few weeks), aphasia, if the right side be paralyzed (right lateral H.), and with little or no anæsthesia,

[1] Henry D. Noyes, "Acute Myelitis with Double Optic Neuritis." Knapp's Archives of Ophthalmology, IX., p. 199, 1880.

is quite certainly due to an extensive superficial lesion of the area supplied by the middle cerebral artery. We would expect to find, as in Westphal's case,[1] softening of the motor zone and of the gyri lying at the extremity of the fissure of Sylvius, viz., the inferior parietal lobule, the supra-marginal gyrus, the gyrus angularis, and the subjacent white substance. Embolism or thrombosis of the Sylvian artery would be the most likely pathological cause of the softening.

d. Lateral H. with moderate loss of power in one-half of the body, especially if associated with impairment or loss of muscular sense, would probably be due to a lesion of the inferior parietal lobule and the gyrus angularis with their subjacent white substance, penetrating deeply enough to sever or compress the optic fasciculus on its way caudad from the primary optic centre to the visual half-centre (the cuneus opposite to the dark half-field).

In all the above symptom-groups of the second category there would be full pupillary reaction and no atrophy of the optic nerves, unless choked disk has occurred and obscured the phenomena. Even with complicating choked disk, there should not be hemiopic pupillary inaction previous to the setting in of atrophy.

e. Lateral H. with other symptoms indicating lesion of the primary optic centres on one side (viz., the corpus geniculatum laterale and one lobus opticus) should be here considered, but our present knowledge is not such as to enable us to recognize disease thus situated. There would certainly be, besides lateral H., hemiopic pupillary inaction, and early atrophy of the optic nerves, as the primary optic centres contain a centre for reflex movements and a trophic centre for the optic nerves. We might theoretically suggest that there would be disorders of sensibility, and perhaps choreic-ataxic movements of the limbs on the same side as the darkhalf-fields.

f. Lateral H. with hemiopic pupillary inaction and with

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[1] C. Westphal: "Zur Localisation der Hemianopsie und des Muskelgefühls beim Menschen." Charité-Annalen, viii., p. 466, 1882.

paralytic symptoms belonging to the class known as crossed paralysis would indicate a lesion at the base of the brain, upon the crus cerebri, injuring the tractus opticus opposite to the dark half-fields, and adjacent structures. The most probable combination would be paralysis of the third, fourth, and sixth cranial nerves on the side of the normal half-fields (one or all of these nerves); with partial hemiplegia, without anæsthesia, on the same side as the dark half-fields, caused by injury to the crus cerebri. An extensive lesion thus placed might also affect the trigeminus. In a fully developed case of this group, we would have as direct symptoms paralysis of the ocular nerves, and perhaps of the trigeminus; as crossed symptoms, lateral hemianopsia, with hemiopic pupillary inaction and early atrophy of the optic nerves; also partial paralysis without anæsthesia on the same side of the body as the dark half-fields. In some cases, choked disks would appear early without interfering with the hemiopic symptoms; at least not until general atrophy of both optic nerves and complete blindness set in.

II.—Heteronymous H. with other symptoms of disease at the base of the brain.

a. Bi-nasal H. with paralysis of all, or nearly all, of the motor and sensory nerves of both eyes, would indicate a lesion inclosing the chiasm, spread out laterally from it so as to affect the orbital nerves, leaving the central (decussating) fibres of the chiasm intact.

b. Bi-temporal H. might be associated with unilateral or bilateral anosmia, thus indicating disease in the mediofrontal edge of the chiasm, extending frontad upon the orbital roof.

c. Bi-temporal H. in one eye, with complete blindness of the other, and paralysis of some or of all the nerves which enter the orbit of the blind eye would indicate a lesion laterad of the chiasm involving the orbital nerves, the optic nerve, and three-quarters of the chiasm (as represented in the diagram of Case I.).

In symptom-groups *a* and *b* there would be hemiopic pupillary inaction, with partial atrophy of both optic

nerves; the associated or consensual pupillary reaction, as well as accommodative pupillary contraction, might be preserved.

In symptom-group *c* there would be hemiopic pupillary inaction in one eye, and total loss of direct pupillary reflex in the other (blind eye). Consensual pupillary contraction would not take place when the light was thrown into the blind eye, but the reverse would be true provided that the motor oculi nerve of the blind eye were not paralyzed.

All these symptom-groups, again, may be complicated with choked disk, though this rarely occurs.

Lastly, more irregular grouping of symptoms of this category, not explicable by any of the above diagnostic propositions, would quite certainly be due to the presence of multiple lesions (in syphilitic subjects especially).